

# Characterizing the association between parenting and adolescent social phobia

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## Abstract

**Objectives:** For characterizing the association between parenting and offspring social phobia (SP), contrasting maternal vs. paternal contributions, putative predictors of unfavorable parenting behaviors and its specificity for SP are warranted to delineate targeted prevention and intervention strategies.

**Methods:** A population-based sample of 1053 adolescents was followed-up using the M-CIDI. Parenting was assessed via questionnaire in offspring passing the high risk period for SP-onset. Natal complications and childhood serious health problems as assessed by maternal reports were hypothesized to relate to unfavorable parenting.

**Results:** The pattern of maternal overprotection, paternal rejection and lower emotional warmth was associated with SP, but not with other offspring anxiety disorders. Natal complications were related to overprotection and lower emotional warmth; trend-level associations emerged for serious health problems and unfavorable parenting.

**Conclusions:** Paternal behavior appears particularly relevant for SP. The pattern of maternal overprotection, paternal rejection and lower emotional warmth was observed in SP only, suggesting that its detailed assessment provides a promising opportunity for targeted prevention and intervention in SP.

**Keywords:** Social phobia Anxiety disorders Parenting Rearing Specificity Development

## 1. Introduction

Familial factors such as parental psychopathology and family environment have been demonstrated to play a prominent role for the onset and course of social phobia (SP) (Fyer, Mannuzza, Chapman, Liebowitz, & Klein, 1993; Knappe, Beesdo-Baum, & Wittchen, 2010; Lieb, Wittchen, et al., 2000; McClure, Brennan, Hammen, & Le Brocque, 2001; Merikangas, Lieb, Wittchen, & Avenevoli, 2003). Nonetheless, associations of parental psychopathology with offspring SP as well as with other offspring disorders indicated low to modest specificity of the parent–offspring-transmission of SP. These disparate findings suggest that parental psychopathology may generally predispose to susceptibility for the onset of different types of

psychopathology in offspring including SP (Knappe et al., 2010). Thus, other factors, i.e., family environment, may help to identify which offspring are likely to develop SP rather than a different anxiety disorder. As general dimensions of parenting such as rejection and control appear to account only for a limited amount of variance in childhood anxiety (McLeod, Wood, & Weisz, 2007), identification of specific subdimensions, along with putative moderators and mediators of unfavorable parenting is warranted to inform theory development and future research. A deeper understanding of the relationship between parenting and offspring SP could provide useful information for the early diagnosis of (social) anxiety and targeted intervention with parents in altering potentially anxiety-inducing rearing practices.

Parenting behavior has been particularly emphasized as a putative risk factor for offspring SP. Research from both clinical and population-based samples yielded strong positive associations of unfavorable parenting behaviors such as lower levels of emotional warmth, and higher levels of overprotection and rejection with offspring SP (Bögels, van Oosten, Muris, & Smulders, 2001; Knappe, Beesdo, Fehm, Höfler, et al., 2009; Knappe, Lieb, et al., 2009). Notably, a comprehensive theory on how unfavorable parenting contributes to the onset of offspring SP in particular or psychopathology in general does not yet exist (O'Connor, 2002) and underlying mechanisms that promote SP in the offspring remain speculative. Studies so far have focused on parents' modelling of anxious behavior (i.e., Gruener, Muris, & Merckelbach, 1999; Muris, Steerneman, Merckelbach, & Meesters, 1996), parental attitudes and actions (Bögels, van Dongen, & Muris, 2003; Turner, Beidel, Roberson-Nay, & Tervo, 2003; Whaley, Pinto, & Sigman, 1999), and verbal and non-verbal behavior towards their offspring (Creswell et al., 2008; Harvey, Ehlers, & Clark, 2005; Murray, Cooper, Creswell, Schofield, & Sack, 2007). Parental overprotection or lack of emotional warmth are suggested to prevent offspring from exposure to social situations and acquiring social skills (Rapee & Spence, 2004), and to increase offsprings' dependence upon their parents (Wood, McLeod, Sigman, Hwang, & Chu, 2003). In turn, unfavorable parenting likely reinforces offspring's anxiety (Rubin, Hastings, Stewart, Henderson, & Chen, 1997) and hampers the development of control, mastery and autonomy (Rapee & Spence, 2004; Wood et al., 2003). Despite convincing evidence for an association between these parenting dimensions and offspring SP, details of this relationship regarding the role of mothers and fathers, origins of specific parenting behaviors and the specificity for SP remain unclear.

First, discussion surrounds the question on unique contributions of maternal and paternal behaviors to offspring SP: Some studies (Bögels & van Melick, 2004; Heider et al., 2008; Hudson & Rapee, 2002; Lindhout et al., 2006; McLeod et al., 2007; van Brakel, Muris, Bögels, & Thomassen, 2006) found that maternal and paternal behaviors are similarly associated with offspring anxiety. In contrast, others indicate differences between mothers and fathers, favoring a dominant role of the father (Bögels & Perotti, 2011; Bögels & Phares, 2008). While mothers are supposed to teach social wariness in their low socially anxious offspring, fathers may serve as an important role model on coping with fears and signalling that the (social) world can be mastered in their high socially anxious offspring (Bögels, Stevens, & Majdandzic, 2011). One parent may reinforce or even compensate for the unfavorable behavior of the other (Bögels & Brechman-Toussaint, 2006; Bögels & Phares, 2008).

Second, identification of the origins and predictors of (unfavorable) parenting behavior is still sparse: While some found unfavorable parenting to be more pronounced in offspring of psychopathologically affected parents (Lindhout et al., 2006; Woodruff-Borden, Morrow, Bourland, & Cambron, 2002), others reported that parental psychopathology and parenting

are unrelated (Bögels, Bamelis, & van der Bruggen, 2008) but may interact on the risk for offspring SP (Knappe, Lieb, et al., 2009). Further, parenting behavior may be derived from the so-called vulnerable child syndrome. That is, parents who view their offspring as more vulnerable or fragile due to natal complications such as low birth weight, preterm birth or delivery, or serious (life-threatening) health problems may elicit higher levels of parental protection (Thomasgard, Shonkoff, Metz, & Edelbrock, 1995) or emotional warmth (Hayward et al., 2008). Such parenting, in turn, may prevent the child from engaging in social interactions, promote offsprings' view of the world as unsafe or threatening (Freemann Duncan & Caughy, 2009; Green & Solnit, 1964), and foster social anxiety (or SP), unless the other parent compensates for the unfavorable parenting (i.e., in showing the opposite parenting behavior). We may further speculate that particular paternal behavior may be relevant for SP (Bögels et al., 2011), i.e., when fathers react with less warmth or even rejection towards their fragile offspring or as a contrast to the close mother–offspring dyad. Thus, to better understand the relationship between parenting and offspring SP, distal variables may be considered as putative predictors of unfavorable parenting, even when they do not necessarily directly relate to offspring SP or anxiety per se (Bandelow et al., 2004; Berle, Mykletun, Daltveit, Rasmussen, & Dahl, 2006; Betts, Williams, Najman, & Alati, 2011; Mallen, Mottram, & Thomas, 2008).

Finally, comparative research to date is mostly based on a single or limited set of parenting behaviors that reveal few differences across the anxiety disorders. For example, SP-patients reported higher levels of parental rejection and control than panic disorder patients (Rapee, 1997; Rapee & Melville, 1997), and patients with panic disorder or obsessive–compulsive disorder (OCD) rated their parents as more protective than non-anxious controls (Turgeon, O'Connor, Marchand, & Freeston, 2002). Given the high comorbidity among offspring anxiety disorders, it remains however unclear which unfavorable parenting dimensions are linked to SP particularly or to anxiety disorders generally.

Since unfavorable parenting may serve as a risk factor for the onset and course of SP, and as a starting point for family-based early prevention and targeted intervention (Ginsburg & Schlossberg, 2002; Knappe et al., 2010), we chose to examine whether (1) maternal and paternal parenting behaviors are differentially related to offspring SP, and hypothesize (2) that natal complications and serious health problems are putative predictors of unfavorable parenting (i.e., higher levels of overprotection, lower levels of emotional warmth). Finally, (3) associations of parenting behaviors with other anxiety disorders in the offspring are tested to consider the specificity of these factors for SP. Parental psychopathology will be considered as a covariate in all analyses. Data stem from a community sample of adolescents and young adults that was prospectively followed-up across the core high risk period for SP onset up to the third decade of life.

## **2. Methods**

### *2.1. Sample and study design*

Data were collected as part of the prospective longitudinal Early Developmental Stages of Psychopathology (EDSP)-Study. The study was designed as a random regional representative population sample of a German community in the metropolitan area of Munich to study the natural course of early stages of mental disorders and to identify risk factors for their onset and course. The study includes follow-up surveys (T1, T2, T3), a family history component (T0, T2, T3) and direct assessment of parents (T1, T3). The EDSP project and its family

genetic supplement have been approved by the Ethics Committee of the Medical Faculty of the Technische Universität Dresden (No: EK-13811). All participants (in cases of aged 18 or younger the parents) provided written informed consent. Detailed descriptions of the EDSP design and field procedures are reported elsewhere (Lieb, Isensee, Von Sydow, & Wittchen, 2000; Wittchen, Perkonig, Lachner, & Nelson, 1998).

Briefly, the EDSP-study consists of a baseline survey conducted in 1995 (T0) with N = 3021 individuals (response rate (RR) 71%) of a younger (N = 1395; aged 14–17 years at baseline) and an older study cohort (N = 1626; 18–24 years at baseline). The first followup (T1, range 1.2–2.1 years since baseline) was conducted only for the younger cohort (N = 1228; RR = 87.8%), whereas the second (T2, range 2.8–4.1 years after baseline; N = 2548; RR = 84.3%) and third follow-up (T3, range 7.3–10.6 years after baseline; N = 2210; RR = 73%) were conducted among all subjects. There was no selective drop out (attrition) from baseline to 10-year follow-up for participants with SP or other anxiety disorders (OR range 0.8–1.4,  $p > .05$ ).

## *2.2. Diagnostic assessment*

### *2.2.1. Assessment in respondents (offspring)*

Mental disorders were assessed face to face by trained interviewers with the computer-assisted version of the Munich- Composite International Diagnostic Interview (DIA-X/M-CIDI) (Wittchen & Pfister, 1997), an updated version of the World Health Organization's CIDI version 1.2 (WHO, 1990, 1992). Most interviewers were psychologists in postgraduate training for psychotherapy or full-time professional health research interviewer who all received 2 full weeks of CIDI-training, followed by at least 10 practice interviews that were closely supervised (Wittchen, Perkonig, et al., 1998).

The DIA-X/M-CIDI allows for the standardized assessment of symptoms, syndromes and diagnoses of 48 mental disorders according to DSM-IV and ICD-10 criteria along with information about onset, duration, and severity. All diagnoses are based on the DSM-IV/DIA-X algorithms. Reliability and validity are moderate to good for all the disorders covered by the DIA-X/M-CIDI. Kappa for diagnostic test–retest reliability was 0.72 for DSM-IV SP (Wittchen, Lachner, Wunderlich, & Pfister, 1998). Validity of the DSM-IV SP diagnoses compared with independent clinical consensus diagnoses by treating physicians was estimated with a kappa of 0.80 (Reed et al., 1998).

At baseline, the DIA-X/M-CIDI was used to assess lifetime diagnoses. The follow-up surveys administered a modified version of the DIA-X/M-CIDI that covered the time interval since the last interview. To increase validity, response lists regarding possible socially distressing situations and a list of social fear symptoms are used (Wittchen, Kessler, & Üstün, 2001). Rates for lifetime anxiety disorders in offspring ranged between 2.47% for panic disorder and 22.0% for specific phobia in the total sample (N = 1395) and between 2.6% for generalized anxiety disorder and 21.95% for specific phobia in the study sample (N = 1053/1395, see below). Respective rates for SP were 7.48% (N = 106/1395) and 7.41% (N = 82/1053).

### *2.2.2. Assessment in parents*

In accordance to the EDSP analyses conventions, diagnosis of parental psychopathology (SP, other anxiety, depressive or substance use disorders either in mother or father; lifetime) was derived by aggregation of diagnostic information from direct DIA-X/M-CIDI interviews in parents (at T1, T3; N = 1152 mothers, N = 211 fathers) and indirect family history

information using the respondents as informants (at T3, T2, T0; overall N = 3021). A priority hierarchy to aggregate information on parental psychopathology was derived after examination of agreement patterns between family history reports and available parent interview data (Beesdo, Pine, Lieb, & Wittchen, 2010). That is, parents' direct information was considered most reliable and was used when available. Interviews mostly took place in the parents' homes, separate from their offspring and were conducted by trained clinical interviewers who were blind to the diagnostic findings of offspring. When direct information was not available, T3 family history information was used, followed by T2 and T0, respectively, because greater agreement was found for respondents at higher ages (T3: 21–34 years, T2: 17–28 years, T0: 14–24 years). Indirect parental diagnoses were derived from family history data collected with offspring as informants using the M-CIDI family history module using a modified version of the Family History Research Diagnostic Criteria (Andreasen, Endicott, Spitzer, & Winokur, 1977). At baseline, offspring were asked M-CIDI questions to assess the key symptoms of parental DSM-IV disorders and whether their parent sought professional help because of his or her respective symptoms. At T2 and T3, an extended version of the family history module was used, containing fully structured sections covering M-CIDI/DSM-IV criteria. Referring to the younger cohort sample, N = 589/1395 (41.3%) parents had an anxiety disorder (among those are N = 156/1395 (10.9%) with SP), N = 452/1395 (32.1%) and N = 164/1395 (12.2%) had depressive or alcohol use disorders respectively. N = 855/1395 (60.4%) met criteria for any of these diagnoses. When no indications for parental diagnoses from any of the two sources were given, parents were classified to have “no diagnosis”.

### *2.3. Assessment of parenting*

The German version of the Questionnaire of Recalled Parental Rearing Behavior (FEE, (Schumacher, Eisemann, & Brähler, 1999) is based on the Swedish EMBU (“Egna Minnen Beträffande Uppfostran”, Perris, Jacobsson, Lindström, von Knorring, & Perris, 1980), and was administered to offspring at T1. Each of the three FEE scales consists of 8 items, according to which offspring characterize experiences of rejection (i.e., “Did your parents punish you even for small things?”), emotional warmth (“Did your parents show their affection to you when others were around?”), and overprotection (“Did your parents sometimes not allow you to do things others at your age were allowed to do because they were afraid something could happen to you?”), separately for mothers and fathers on a four-point Likert scale (1 – no, never to 4 – yes, always). Analog to previous reports (Knappe, Beesdo, Fehm, Lieb, & Wittchen, 2009; Knappe, Lieb, et al., 2009), we also calculated a mean score across maternal and paternal rearing for each FEE scale to indicate overall parenting. Scores for each scale ranged from 8 to 32, with higher scores on the rejection and overprotection scale, and lower scores on the emotional warmth scale indicating more unfavorable parenting. Reliability and validity of the FEE were reported to be high (Arrindell et al., 1994; Schumacher et al., 1999). Chronbachs alpha was .69, .75, and .85 for paternal and maternal overprotection, rejection, and emotional warmth, respectively (Knappe, Beesdo, Fehm, Höfler, et al., 2009).

### *2.4. Assessment of natal complications and serious health problems*

Parents (predominantly mothers N = 1026; fathers N = 27) were asked to recall pre-, perinatal and neonatal complications at T1. To increase validity, response lists for each of these categories were used. Prenatal complications include roseola (German measles), anemia, infections with high fever and need of antibiotica, infections of kidney or bladder (gestosis), blood poisoning, high blood pressure, bleeding, excessive nausea, abortion attempt. Perinatal

complications include dystocia, complications with umbilical cord, unfavorable fetal lie, blood loss during delivery, amniorrhexis, forceps delivery, unplanned caesarian section. Neonatal complications include jaundice, blood group incompatibility, respiration, convulsions, birth injury, low birth weight, premature birth, infections or APGAR  $\leq 7$ . Based on independent ratings of 5 experts (3 psychiatrists, 1 pediatricist, 1 gynecologist), a natal risk index to describe the proportion of natal complications was created (see Wittchen, Lieb, Schuster, & Oldehinkel, 1999 for further details): No or low natal risk was indicated when no complications or only umbilical or inguinal hernia were present (unweighted N = 411/1053; weighted 38.6%). Medium natal risk index was indicated when at least one natal complication (e.g. unfavorable fetal lie, unplanned caesarian section) categorized as moderate by the experts was present (N = 316/1053; 31.2%). A high natal risk referred to the presence of any severe natal complications (e.g. abortion attempt, APGAR  $\leq 7$ ; N = 326/1053; 30.2%).

In addition, mothers were asked whether their child was ever hospitalized for one week or longer, or whether their child ever had a life-threatening emergency that required stationary admission from birth until kindergarten age. This item was used as a proxy indicator for the presence of serious health problems (no health problems: N = 797/1053; 75.3%; only once: N = 171/1053, 16.5%; two times or more: N = 85/1053; 8.2%).

### *2.5. Statistical analysis*

Results (% , means, standard deviations, coefficients) were weighted by age, gender, and geographic location at baseline to match the distribution of the original sampling frame (Lieb, Wittchen, et al., 2000; frequencies (Ns) are reported unweighted. The Stata Software package 11.0 (StataCorp, 2010) was used to compute robust variances, confidence intervals, and p-values (by applying the Huber–White sandwich matrix) which is required when analyses are based on weighted data (Royall, 1986). No adjustment for multiple testing was applied, because the individual tests were related to individual hypotheses and adjustment would treat them as reflecting a global hypothesis which is questionable in substantive terms (Savitz & Olshan, 1995).

Logistic regressions provided odds ratios (OR) for the bivariate outcome of offspring SP and other offspring anxiety disorders, respectively. The area under the Receiver Operating Characteristic (ROC) Curve (AUC) was used to quantify the predictive value of logistic regression models. AUC is the probability that a randomly selected outcome case (i.e., SP in offspring) has a higher predicted probability to be a case than a randomly selection non-case (i.e., non-SP in offspring). It ranges from 0.5 (chance level) to 1 (perfect prediction). Univariate linear regression analyses were used to obtain standardized beta coefficients of parenting as an outcome variable, controlled for offspring age, gender and parental psychopathology. Scores of the FEE scales were standardized such that the coefficient (Beta) for each variable reveals the factors by which parenting changes for each increase or decrease of one unit in the independent variable. Betas were tested for significance via t-tests (Pedhazur & Schmelkin, 1991). Testing for normal distribution did not indicate violation of linearity assumptions.

To examine interactions between natal complications (serious health problems) with parenting on the incidence of SP in the offspring, natal complications (serious health problems) were entered as the first predictor into regression analyses, and the unstandardized sum score of the respective parenting scale was entered as the second predictor. For the interaction term as the third predictor, a product term (Aiken & West, 1991) of natal complications (serious health problems) and the standardized sum score of the respective parenting scale were entered in

the regression. To prevent the occurrence of small cell sizes and to increase statistical power of interaction analyses, natal complications were dichotomized into no/low vs. medium/high natal risk (N = 411 vs. 642; 38.6% vs. 61.4%), and serious health problems into none vs. at least one serious health problem (N = 797 vs. 256; 75.3% vs. 24.7%), respectively.

For the current study, we analyzed data from the younger cohort sample for whom data about parenting, natal complications and serious health problems were available (study sample N = 1053/1395).

### **3. Results**

#### *3.1. Associations between maternal and paternal parenting and offspring SP*

Unique contributions of maternal and paternal parenting to the risk of offspring SP were examined in separate regression models for each of the FEE scales, with maternal behavior, paternal behavior and their interaction as independent variables (Table 1). Maternal but not paternal overprotection was associated with offspring SP. In contrast, paternal rejection and lack of emotional warmth but not maternal rejection or emotional warmth were associated with offspring SP. None of these results changed after controlling for parental SP and other parental anxiety, depressive or substance use disorders. AUC was .676 for overprotection, and .660 and .643 for rejection and emotional warmth, respectively.

Analyses were also run separately for female and male offspring (not shown in table). Paternal rejection was associated with SP in both females (OR = 1.18, 95%CI: 1.02–1.36) and males (OR = 1.28, 95%CI: 1.06–1.55). For overprotection, no associations with SP in the subsamples were found. Lower paternal emotional warmth was related to SP in females (OR = 0.89, 95%CI: 0.82–0.97), but only when maternal emotional warmth was also low (interaction: OR = 1.31, 95%CI: 1.07–1.61). Again, results did not change after controlling for parental psychopathology. In males, AUC was .711 for overprotection, and .681 and .653 for rejection and emotional warmth, respectively. In females, AUC was .627 for overprotection, and .671 and .660 for rejection and emotional warmth, respectively.

#### *3.2. Associations of natal complications and serious health problems with offspring SP*

Rates of offspring SP apparently increased with the degree of natal complications (Table 2, upper part). The risk for offspring SP in respondents with medium (p = .078) or high natal risk index (p = .057) was however not substantially higher than in respondents with no/low natal risk index. Regarding serious health problems (Table 2, lower part), offspring with two or more illness events were not more frequently affected by SP than offspring without such events (p = .078). AUC was .633 and .644 for natal risk index and serious health problems as putative predictors for offspring SP, respectively.

#### *3.3. Associations of natal complications and serious health problems with parenting*

Table 3 shows associations of natal complications and serious health problems with parenting in the study sample: Medium and high natal risk were associated with higher levels of maternal overprotection, lower levels of paternal rejection and emotional warmth, as compared to respondents with no/low natal risk index. Notably, for overall parenting (i.e., mean score across maternal and paternal rearing) no associations with natal complications were found (not in table).

The association between two or more serious health problems with higher levels of maternal ( $p = .080$ ) and paternal overprotection ( $p = .064$ ) was not significant. The presence of one serious health problem was significantly associated with lower levels of paternal emotional warmth ( $p = .009$ ), but only a trend level finding emerged with lower levels of maternal emotional warmth,  $p = .077$ .

We finally examined whether natal complications and serious health problems, respectively, interact with parenting on the risk for offspring SP. An interaction would indicate that the combination of natal complications or serious health problems with parenting is superior to one of these conditions alone. Overall, we tested 18 possible interactions (2 factors [natal complications or serious health problems]  $\times$  3 parenting scales [overprotection, rejection, emotional warmth]  $\times$  3 parent conditions [mother, father, both parents]). Among these, no significant interactions were found.

Briefly, natal complications and serious health problems were not substantially associated with offspring SP. Natal complications were however related to higher levels of maternal overprotection, and lower levels of paternal rejection and emotional warmth.

### *3.4. Specificity of the associations between parenting and offspring psychopathology*

To examine whether the association of parenting (overprotection, rejection, emotional warmth) and offspring psychopathology is limited to SP, also specific phobias, panic disorder, agoraphobia, generalized anxiety disorder and OCD were considered as offspring outcomes. Crude associations (controlled only for offspring age and gender) indicated that unfavorable parenting was related to all anxiety disorders, except for OCD. We then controlled for offspring SP due to its high comorbidity with other anxiety disorders, resulting in attenuation of some associations: Maternal rejection and overprotection were positively associated with specific phobias, panic disorder and agoraphobia in offspring. Paternal overprotection was associated with offspring specific phobias, panic disorder, agoraphobia and generalized anxiety disorder. Lower paternal emotional warmth was only associated with offspring panic disorder. Due to the substantial familial aggregation of mental disorders, we additionally controlled for parental psychopathology (including parental SP, other anxiety, depressive and substance use disorders), but findings did not change substantially. Lastly (Table 4), natal complications and serious health problems were taken into account as they emerged as putative predictors of unfavorable parenting: Maternal overprotection remained associated only with specific phobias ( $OR = 1.25$ , 95%CI: 1.06–1.46) and panic disorder ( $OR = 1.80$ , 95%CI: 1.31–2.47). Paternal emotional warmth was unrelated to any of the other anxiety disorders. AUC for the latter regression model ranged between .676 and .708 for SP, and between .646 and .661 for any anxiety disorder.

In sum, parenting was associated with most of the anxiety disorders considered. The particular constellation of maternal overprotection, and paternal rejection and lack of emotional warmth emerged however only for offspring SP. Specifically, associations with lower paternal emotional warmth appeared to be limited to offspring SP.

## **4. Discussion**

The present study aimed to characterize the relationship between parenting and offspring SP. Analyses revealed different contributions of maternal and paternal parenting behaviors to



offspring SP, as well as associations between natal complications and unfavorable parenting. Notably, the constellation of maternal overprotection, paternal rejection and lack of emotional warmth appeared to be specific for SP, with some differences between boys and girls.

Concordant with prior studies (Caster, Inderbitzen, & Hope, 1999; Knappe, Beesdo, Fehm, Höfler, et al., 2009; Knappe, Lieb, et al., 2009; Lieb, Wittchen, et al., 2000; McClure et al., 2001; Rapee & Spence, 2004; Woodruff-Borden et al., 2002), parenting was strongly associated with offspring SP. Our study may help to substantiate these findings in two respects: Contrasting between mothers and fathers revealed that maternal (but not paternal) overprotection, and paternal (but not maternal) rejection and lower emotional warmth were related to offspring SP. Further, rejection appears to be similarly relevant in both sons and daughters, while lower paternal emotional warmth was more relevant in daughters. Findings thus suggest different contributions of mothers and fathers, tentatively supporting recent reports from a Dutch work group, concluding that fathers' behavior has a greater impact on high socially anxious children's anxiety than mothers' behavior (Bögels et al., 2011). Mothers are usually rated as more affectionate (i.e., emotional warm) than fathers (Gerlsma & Emmelkamp, 1994), and are considered to teach social wariness to their low socially anxious children. In offspring with SP however, particularly lack of fathers' emotional warmth along with higher rejection (cf. Bögels et al., 2008) may create uncertainty in offspring and hamper development of social confidence. The however unexpected association between natal complications and lower levels of paternal rejection requires further research.

So far, origins of unfavorable parenting however remained unclear, as parental psychopathology was unrelated to the association of parenting with offspring SP in our study (Knappe, Lieb, et al., 2009). Referring to the vulnerable child syndrome (Freemantle, Duncan & Caughy, 2009; Green & Solnit, 1964) we therefore examined whether parents' tendency towards overly protective behavior in offspring with SP was driven by natal complications or serious health problems: As hypothesized, medium and high natal risk index were associated with higher levels of maternal overprotection and lower levels of paternal emotional warmth, as compared to respondents with no/low natal risk index. Findings on associations with early adversities probably suggest specific social-cognitive processes in parents (O'Connor, 2002). That is, we may provisionally interpret these findings in favor of a vulnerable child syndrome in offspring with SP, suggesting that if offspring are (perceived as) more fragile, i.e., due to natal complications or serious health problems (by their parents), mothers exhibit higher levels of protection: Already since the very beginning of the mother-offspring relationship, the mother may view her offspring as fragile and in need for protection, especially if she has an (anxiety) disorder herself. Maternal overprotection may prevent the child from potentially dangerous situations in general, but also reflects the lack of maternal encouragement to engage in social situations. Fathers may react with negative or even hostile behaviors, perhaps as a reflection of the father's withdrawal from their fragile offspring or as a response to the close mother-offspring dyad. Despite the intuitive appeal to conclude that parenting behaviors result from parents' cognitions, more specific examination of parents' beliefs about their offspring is warranted for more reliable conclusions particularly in light of the lack of an interaction of early adversities and specific maternal and paternal parenting behaviors on offspring SP. Also, as we did not find early adversities to predict higher levels of parental rejection, and serious health problems did not emerge to contribute to parenting, further variables should be taken into account.

Interestingly, the constellation of maternal overprotection, and paternal rejection and lower emotional warmth was found in offspring SP only, also after controlling for natal complications and serious health problems as putative predictors of unfavorable parenting.

This particular constellation of behaviors may represent a malleable model of behavior in social interactions, resulting in offspring SP. Again it becomes apparent that mothers and fathers play different roles for their offspring, and that the combination of unfavorable maternal and paternal parenting is associated with offspring SP. In fact, most socially phobic offspring (but not offspring with other anxiety disorders) report unfavorable parenting on all three rearing-scales. Moderate AUC estimates however reflect the limited amount of variance explained by our model. Interestingly, AUC for SP descriptively increases when parenting is considered separately for mothers and fathers, and when also distal factors such as natal complications are taken into account. Referencing our findings to those from other research is notably challenging. To our knowledge, this is the first community-based study directly comparing associations of parenting with SP to other anxiety disorders in adolescent offspring. Probably due to the high comorbidity and because studies diverge in assessment tools as well as in considering broad (i.e., more general) dimensions of parenting vs. particular parenting practices, evidence from direct comparisons among subjects with other anxiety or depressive disorders is limited (Alnaes & Torgersen, 1999; Mellon & Moutavelis, 2011; Stark, Humphrey, Crook, & Lewis, 1990). Based upon prior studies in clinical and convenience samples, Rapee (1997) concluded that broad dimensions of parenting such as control and rejection may be specifically associated with anxiety and depression, respectively. Similarly to our study, Heider et al. (2008) found care and overprotection to be associated with SP, specific phobias, generalized anxiety and panic disorder. Again, also Heider et al. did not control associations for comorbidity, so our interpretation in terms of probable specificity of the relationship between parenting and offspring SP remains preliminary, calling for replication and further comparisons with offspring depressive and substance use disorders. For example, Beesdo et al. (2010) compared associations with parenting between generalized anxiety only, other anxiety disorders (excl. comorbid GAD cases), depressive disorders and both anxiety and depressive disorders. Concordant to Rapee, they found parental rejection related to anxiety and depressive disorders (either alone or combined) and overprotection (similar to parental control) related to generalized anxiety and other anxiety disorders as well. Notably, emotional warmth was related to pure depression only pointing to the need to consider also offspring depressive disorders at least as a covariate for emotional warmth, but probably also for parental rejection (as found by Rapee, 1997). More specifically, Mellon and Moutavelis (2011) recently observed differences in the magnitude of correlations between particular parental educational practices and specific offspring anxiety disorders, with parental aversive control and non-responsiveness most strongly related to panic disorder/agoraphobia and generalized anxiety disorder, and medium correlations ( $r$  range between 0.17 and 0.25) to social anxiety. Moreover, findings from Beesdo et al. (2010) evidence that even non-depression-comorbid social anxiety and panic disorder are associated with parental overprotection. Overall, these findings argue for specificity of parenting-offspring-associations, which are probably more reliably to observe when specific parenting behaviors are assessed.

Some limitations need to be mentioned. First, parenting in our study refers to parental overprotection, rejection and emotional warmth. Other parenting behaviors such as intrusiveness or communication styles were not considered here. The term ‘unfavorable’ was used to indicate parenting that confers risk for offspring to develop SP. Also, a diagnostic threshold to define when parenting becomes unfavorable has not yet been established. Secondly, offsprings’ reports on parenting may be driven by offsprings’ temperament (Manassis & Bradley, 1994; Rubin, Nelson, Hastings, & Asendorpf, 1999), by interpersonal processes between parents and offspring (Christensen, Stein, & Means-Christensen, 2003; Heerey & Kring, 2007), attributional bias due to offspring psychopathology (Alfano, Beidel, & Turner, 2006; Wilson & Rapee, 2005), or offsprings’ current mental state. The latter was

however disproved by Gerlsma, Kramer, Scholing, & Emmelkamp (1994). Third, other relevant (natal) factors such as age of mother at childbirth or parity were not considered, and they are likely proxy indicators of underlying associations of early adversities with parenting and offspring SP. Also parents themselves, i.e., their temperamental style, their own rearing histories, their beliefs about the offsprings' temperament (Rubin et al., 1999) may help to further elucidate predictors of (unfavorable) parenting, and to illustrate the likely interactive and interdependent parent–offspring-relationships. Then moderate AUC rates of our study would probably increase. Lastly, associations are correlational and do not allow for any causal or temporal interpretation. We decided to consider logistic regressions between variables instead of presuming temporal relationships between the variables, i.e., moderators/mediators as claimed by Kraemer et al. (1997). For example, early adversities may be considered as a putative moderator of the association between parenting and offspring SP. However, offspring SP (and parental psychopathology as well) refers to lifetime diagnostic status, parenting relates to current rearing experiences in offspring assessed at T1.

With these limitations in mind, but also acknowledging direct assessment of natal complications in mothers by using response lists to facilitate recall and the fairly recent collection of parenting while most of offspring lived with their parents, findings suggest a probable rational for the development of SP, which needs to be examined in greater detail with regard to its temporal and causal pathways using data of high risk birth cohort studies. With regard to its specificity for SP, parenting could be useful indicator for the development of SP in offspring. Albeit causal inferences on the particular role of parenting for the onset and course of SP are precluded (Knappe & Martini, 2010), its detailed assessment may provide a promising intervention target in children and adolescents with SP, particularly when treating younger patients for whom directly administered interventions such as CBT may not always be appropriate.

**Table 1**  
Associations of parenting with offspring social phobia in the total sample (data available from  $N = 1110/1395$ ).

Parenting	Total sample		Offspring unaffected by SP		Offspring affected by SP		Risk for offspring SP			p
	M	SD	M	SD	M	SD	OR	95%CI		
Rejection										
Mother	9.66	2.13	9.61	2.02	10.25	3.06	1.10	0.95	0.95	.188
Father	9.62	2.05	9.53	1.84	10.65	3.59	<b>1.22</b>	<b>1.08</b>	<b>1.38</b>	<b>.001</b>
Mother*father <sup>a</sup>	—	—	—	—	—	—	0.88	0.71	1.08	.213
Overprotection										
Mother	14.03	3.37	13.91	3.29	15.49	3.96	<b>1.11</b>	<b>1.01</b>	<b>1.22</b>	<b>.024</b>
Father	13.00	3.17	12.92	3.06	14.00	4.14	1.04	0.92	1.18	.518
Mother*father <sup>a</sup>	—	—	—	—	—	—	0.97	0.80	1.19	.790
Emotional warmth										
Mother	24.42	4.02	24.47	3.91	23.85	5.11	1.05	0.96	1.15	.312
Father	22.33	4.86	22.45	4.71	20.77	6.24	<b>0.92</b>	<b>0.86</b>	<b>0.99</b>	<b>.027</b>
Mother*Father <sup>a</sup>	—	—	—	—	—	—	<b>1.20</b>	<b>1.00</b>	<b>1.43</b>	<b>.046</b>

Note: SP, social phobia; M, mean; SD, standard deviation, OR, odds ratio, controlled for offspring age and gender, parental anxiety, depressive or substance use disorders; comparison group are respondents without social phobia. CI, confidence interval; bold prints indicate statistical significance at  $p < .05$ .

<sup>a</sup> Indicates interaction effects of maternal and paternal behavior.

**Table 2**  
Associations of natal risk index and serious health problems with offspring social phobia.

	Study sample (N= 1053)		Offspring unaffected by SP (N= 971)		Offspring affected by SP (N= 82)		Risk for offspring SP			
	N	%	N	%	N	%	OR	95%CI	p	
Natal risk index										
No/low	411	38.58	384	39.53	27	26.78	Ref			
Medium	316	31.20	289	30.79	27	36.33	1.72	0.94	3.15	.078
High	326	30.21	298	29.68	28	36.89	1.79	0.98	3.25	.057
Serious health problems										
None	797	75.33	741	76.22	56	64.09	Ref			
One	171	16.33	155	16.15	16	21.3	1.53	0.82	2.85	.179
Two or more	85	8.15	75	7.63	10	14.61	2.03	0.92	4.48	.078

Note: SP, social phobia; Ref, reference group; N, unweighted number; %, weighted percentages. OR, odds ratio, controlled for offspring age and gender, parental anxiety, depressive or substance use disorders. CI, confidence interval.

**Table 3**  
Associations of natal risk index and serious health problems with parenting in the study sample (N= 1053).

	Maternal behavior														
	Rejection					Overprotection					Emotional warmth				
	M	SD	Beta	T	p	M	SD	Beta	T	p	M	SD	Beta	T	p
Natal risk index															
No/low	9.58	1.78	Ref			13.58	13.58	Ref			24.69	3.87	Ref		
Medium	9.70	2.27	0.02	0.61	.542	14.29	3.50	<b>0.10</b>	<b>2.47</b>	<b>.014</b>	24.35	4.17	−0.04	−1.06	.288
High	9.71	2.12	0.02	0.68	.497	14.27	3.46	<b>0.09</b>	<b>2.43</b>	<b>.015</b>	24.59	4.17	−0.01	−0.36	.721
Serious health problems															
None	9.58	1.93	Ref			13.85	3.25	Ref			24.72	3.94	Ref		
One	9.77	2.36	0.03	0.76	.444	14.30	3.53	0.05	1.20	.230	24.01	4.02	−0.06	−1.77	.077
Two or more	10.12	2.33	0.06	1.49	.137	14.84	3.76	0.07	1.75	.080	24.18	4.36	−0.03	−0.78	.436
Paternal behavior															
	Rejection					Overprotection					Emotional warmth				
	M	SD	Beta	T	p	M	SD	Beta	T	p	M	SD	Beta	T	p
Natal risk index															
No/low	9.68	1.99	Ref			12.92	3.12	Ref			22.83	4.65	Ref		
Medium	9.66	2.37	−0.01	−0.15	.882	12.90	3.14	0.00	−0.08	.935	21.98	5.32	<b>−0.08</b>	<b>−0.20</b>	<b>.050</b>
High	9.40	1.57	<b>−0.07</b>	<b>−2.00</b>	<b>.045</b>	13.04	3.17	0.02	0.39	.697	22.40	4.72	−0.04	−1.06	.290
Serious health problems															
None	9.58	2.07	Ref			12.89	3.13	Ref			22.72	4.76	Ref		
One	9.50	1.68	−0.02	−0.70	.485	12.90	3.19	0.00	0.05	.963	21.37	5.22	<b>−0.10</b>	<b>−2.60</b>	<b>.009</b>
Two or more	9.85	2.11	0.03	0.69	.490	13.71	3.10	0.07	1.85	.064	21.93	5.22	−0.03	−0.08	.401

Note: SP, social phobia; Ref, reference group; M, mean; SD, standard deviation; Beta, standardized regression coefficient from linear regression, controlled for age and gender, parental anxiety, depressive or substance use disorders. Bold prints indicate statistical significance at  $p < .05$ .

**Table 4**  
Specificity of associations between parenting and anxiety disorders in offspring in the study sample (N= 1053).

Offspring psychopathology	Maternal behavior											
	Rejection				Overprotection				Emotional warmth			
	OR	95%CI		p	OR	95%CI		p	OR	95%CI		p
Specific phobia (N= 233, 21.9%)	1.14	0.98	1.33	.100	<b>1.25</b>	<b>1.06</b>	<b>1.46</b>	<b>.007</b>	1.03	0.87	1.21	.772
Panic disorder (N= 31, 2.9%)	1.33	0.99	1.78	.055	<b>1.80</b>	<b>1.31</b>	<b>2.47</b>	<b>.000</b>	0.80	0.54	1.17	.250
Agoraphobia (N= 45, 4.51%)	1.28	0.96	1.70	.094	1.34	0.98	1.84	.065	0.98	0.69	1.40	.922
Generalized anxiety disorder (N= 27, 2.6%)	1.04	0.81	1.34	.763	1.06	0.76	1.47	.735	1.00	0.66	1.52	.992
Obsessive compulsive disorder (N= 17, 1.5%)	0.90	0.65	1.26	.547	0.81	0.54	1.22	.311	1.01	0.56	1.82	.969
Any anxiety disorder <sup>a</sup> (N= 402, 38.2%)	1.16	0.97	1.39	.098	<b>1.19</b>	<b>1.01</b>	<b>1.39</b>	<b>.034</b>	1.07	0.91	1.26	.385
Offspring psychopathology												
	Paternal behavior											
	Rejection				Overprotection				Emotional warmth			
	OR	95%CI		p	OR	95%CI		p	OR	95%CI		p
Specific phobia (N= 233, 21.9%)	1.14	0.97	1.33	.109	<b>1.29</b>	<b>1.09</b>	<b>1.52</b>	<b>.003</b>	0.88	0.75	1.03	.110
Panic disorder (N= 31, 2.9%)	0.73	0.44	1.20	.212	<b>1.81</b>	<b>1.26</b>	<b>2.59</b>	<b>.001</b>	0.73	0.52	1.04	.078
Agoraphobia (N= 45, 4.51%)	1.14	0.90	1.45	.277	<b>1.39</b>	<b>1.03</b>	<b>1.89</b>	<b>.033</b>	0.81	0.60	1.09	.166
Generalized anxiety disorder (N= 27, 2.6%)	0.99	0.71	1.37	.942	1.45	0.97	2.15	.068	0.85	0.55	1.31	.459
Obsessive compulsive disorder (N= 17, 1.5%)	1.09	0.79	1.51	.581	1.20	0.86	1.68	.286	0.87	0.64	1.17	.355
Any anxiety disorder <sup>a</sup> (N= 402, 38.2%)	1.07	0.90	1.28	.425	1.09	0.93	1.28	.301	0.91	0.78	1.07	.254
Offspring psychopathology												
	Parental behavior											
	Rejection				Overprotection				Emotional warmth			
	OR	95%CI		p	OR	95%CI		p	OR	95%CI		p
Specific phobia (N= 233, 21.9%)	<b>1.19</b>	<b>1.02</b>	<b>1.40</b>	<b>.028</b>	<b>1.31</b>	<b>1.11</b>	<b>1.55</b>	<b>.001</b>	0.93	0.78	1.09	.367
Panic disorder (N= 31, 2.9%)	1.13	0.81	1.57	.471	<b>2.01</b>	<b>1.41</b>	<b>2.85</b>	<b>.000</b>	0.73	0.51	1.05	.092
Agoraphobia (N= 45, 4.51%)	1.32	1.00	1.74	.051	<b>1.42</b>	<b>1.05</b>	<b>1.93</b>	<b>.023</b>	0.85	0.62	1.16	.292
Generalized anxiety disorder (N= 27, 2.6%)	1.01	0.72	1.40	.976	1.30	0.92	1.83	.138	0.88	0.57	1.35	.553
Obsessive compulsive disorder (N= 17, 1.5%)	1.08	0.74	1.56	.695	1.04	0.76	1.43	.797	0.85	0.56	1.30	.467
Any anxiety disorder <sup>a</sup> (N= 402, 38.2%)	1.16	0.98	1.38	.082	1.15	0.98	1.35	.089	0.98	0.83	1.15	.775

OR, odds ratio, controlled for offspring age, gender, social phobia and parental anxiety, depressive or substance use disorders; natal complications, serious health problems, comparison group are respondents without the respective anxiety disorder. CI, confidence interval; bold prints indicate statistical significance at  $p < .05$ . N, unweighted number, % weighted percentages

<sup>a</sup> Includes social phobia, specific phobia, panic disorder, agoraphobia, generalized anxiety disorder, obsessive-compulsive disorder, phobias NOS.

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